ment of her arteritis with antitumor necrosis factor-α. Antitumor necrosis factor-α inhibits granuloma formation, and there are many reports of tuberculosis after its use.[5-6]

As is the case with most vasculitides, the etiology of Takayasu arteritis is not known. There is considerable overlap with other syndromes of large-vessel arteritis. Not all evidence points toward tuberculosis as a cause, and other pathophysiologic mechanisms have been proposed. Nevertheless, the association between Takayasu arteritis and *Mycobacterium tuberculosis* is an intriguing one and awaits further study.

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Reply

I thank Dr McPhail for his insightful and interesting comments. I would certainly agree with him that we did not address the etiology of Takayasu's arteritis. The focus of the case report was to describe a rare form of graft infection by *Mycobacterium tuberculosis* and its successful treatment as well as briefly summarize the bacterial etiology and treatment of aortic graft infections in general. It was not our intention to discuss the association of Takayasu's arteritis with *M. tuberculosis.*

Dr McPhail has raised some very interesting issues about the etiology of Takayasu's arteritis and the possible connection with tuberculosis. Of course there is a challenge in trying to attribute causation (as with Crohn's disease or sarcoidosis) as opposed to association. The association between Takayasu's arteritis and tuberculosis has been noted and is well referenced by Dr. McPhail. But one third of the world's population is infected with tuberculosis (World Health Organization), and that number is much higher in India and similar places. Again, the association does not ascribe causality: the same immune defect that causes the arteritis may permit activation of latent tuberculosis infection.

To the best of our knowledge *M. tuberculosis* antigens have not been found in the arterial wall of patients with Takayasu's arteritis or other vasculitides. We certainly agree with Dr McPhail and the references provided that in the case we have described, the patient's quiescent tuberculosis was reactivated by infliximab (Remicade, Centiocor, Inc, Malvern, Pa), an anti-tumor necrosis factor-α monoclonal antibody.

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Regarding "Ultrasound findings after radiofrequency ablation of the great saphenous vein"

We would like to comment on the article by Salles-Cunha et al: Ultrasound findings after radiofrequency ablation of the great saphenous vein: Descriptive analysis (J Vasc Surg 2004;40:1166-1173). The development of the radiofrequency (RF) closure technique for treating the incompetent great saphenous vein (GSV) was completed by multiple specialties, with dermatology taking a significant role.[1,2] Dermatologic surgeons were the first to apply tumescence to ambulatory phlebectomy and/or ligation of the GSV and its tributaries.[3,4] We therefore used tumescent anesthesia with radiofrequency or other energies for endoluminal closure/ablation of the GSV. Tumescent anesthesia or the placement of large volumes of dilute anesthesia in a perivascular position serves several purposes: first, to protect perivascular tissues from the thermal effects of intravascular energy such as RF; second, to decrease the diameter of the treated vein to allow for better contact of the RF electrodes with the vein wall, and thus secondarily to reduce intravascular blood for nonspecific coagulation; and third, to provide better and safer anesthesia for patients. All of these effects should serve to reduce perivascular inflammation.

Our initial results showed that tumescent anesthesia to treat the GSV with endovenous techniques resulted in a painless procedure with little down-time and immediate ambulation of the patient.[1,2,5,6] Regarding RF closure, we now have up to 5 years of follow-up on 125 (M.P.G.) and 627 (R.A.W.) patients. We also have treated patients with intravascular lasers, including the 810-nm diode laser, with up to 3 years of follow-up (75 patients, M.P.G.; 36 patients, R.A.W.), and an additional 143 patients with the 1,320-nm intravascular neodymium:yttrium-aluminum-garnet laser with up to 2 years of follow-up (43 patients, M.P.G.; 121 patients, R.A.W.).[7] Our combined clinical experience with endovenous techniques spans 5.5 years, with well over 1,000 patient treatments. Posttreatment duplex evaluation data at regular time intervals exists on the vast majority of these patients.

Contrary to what is reported by Salles-Cunha et al, our experience with tumescent anesthesia used in every one of our patients is a complete lack of small-vessel networks when patients are evaluated postoperatively with duplex ultrasound. We believe that the reason for our failure to find small-vessel networks is not a lack of trying to see them but rather the minimization of inflammation that occurs with tumescent anesthesia placed in the perivascular space during either RF or laser endothelial ablation.

We have not performed ligation of the saphenofemoral junction (SFJ) on any of our over 1,000 patients and question the accuracy of the findings of Salles-Cunha et al, who found a decreased incidence of small-vessel networks in patients whose SFJs were ligated. We suspect that the small number of patients who were treated without ligation of the SFJ (13) vs the 93 patients who did have SFJ ligation produced the false statistical significance of this finding. Alternatively, if inflammation is the most likely cause for small vessel networks, why would ligation decrease the extent or time of inflammation?

Finally, the investigators admit to performing ligation of all tributary veins at the time of ligation at the SFJ. Could it be possible that ligating and disrupting the normal vascular system at the SFJ results in a decrease in the normal number of small-vessel networks and that this accounts for the decrease seen in patients operated on vs patients in whom only the RF is performed?

We are curious about why none of the five studies that we published were not cited in the Salles-Cunha et al publication.[1,2,5-7] We support efforts to share information across special-